

H1N1 influenza and the Australian macroeconomy

George Verikios
Centre of Policy Studies
Monash University

James M. McCaw, Jodie McVernon
Vaccine and Immunisation Research Group
Murdoch Childrens Research Institute and
Melbourne School of Population Health
The University of Melbourne

Anthony H. Harris
Centre for Health Economics
Monash University

Abstract

Early 2009 saw the emergence of an H1N1 influenza epidemic in North America that eventually spread to become the first pandemic of the 21st century. Previous work has suggested that pandemics and near pandemics can have large macroeconomic effects on highly affected regions; here we estimate what those effects might be for Australia. Our analysis applies the MONASH-Health model: a computable general equilibrium model of the Australian economy. We deviate from previous work by incorporating two important short-run mechanisms in our analytical framework: quarterly periodicity and excess capacity. The analysis supports the assertion that an H1N1 epidemic could have significant short-run macroeconomic effects but the size of these effects is highly dependent on the degree of inertia in the markets for physical capital and labour.

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1. Introduction

April 2009 saw the emergence of an H1N1 influenza (swine flu) epidemic in North America that eventually spread to become the first pandemic of the 21st century. By January 2010, H1N1 activity had peaked in most regions of the world but intense pandemic activity was still being observed in North Africa, Southern Asia, and parts of East and Southeast Europe. The pandemic had ended by August 2010, by which time there had been laboratory confirmed cases of swine flu in over 214 countries and, at least, 18,449 deaths (WHO 2010a, 2010b, 2010c).¹ In Australia, there had been 37,562 confirmed cases of swine flu and 191 deaths by the middle of summer (January) 2010, by which time national influenza activity was low (DHA 2010).

A measure of the relative severity of the epidemic in Australia is given by two indicators. Relative to other southern hemisphere countries, Australia's crude rate of confirmed cases per 100,000 population was very high at 166.7; this compares to 74.1 in Chile, 72.8 in New Zealand, and 20.7 in Argentina. Australia also compares unfavourably to northern hemisphere countries on this measure; 21.6 in the UK, 21.1 in Mexico, and 12.5 in the USA. Australia compares more favourably in terms of hospitalisations per 100,000 population at 21.4. Similar rates were observed in New Zealand (22.7), and Argentina (22.1), but there were much lower rates in Chile (9.1), Canada (4.4), the USA (3), and the UK (2.7).² Of the number hospitalisations, 13% were in intensive care units (ICUs) and this was much higher than expected (Bishop 2009). Overall, these measures indicate that with respect to other countries in 2009, Australia's H1N1 epidemic can be regarded as relatively severe. The reported numbers of cases per capita were high, reflecting intensive case finding efforts in the initial phases of the pandemic response, and ready access to laboratory testing resources around the country.

In 2003 there was a near pandemic of SARS (Severe Acute Respiratory Syndrome); analyses of this episode estimated very large temporary economic effects in highly affected Asian regions (Chou et al. 2004; Hai et al. 2004; Lee and McKibbin 2004). This suggests that the 2009 H1N1 pandemic might also have had large temporary economic effects on highly affected regions. Our objective is to estimate what those effects might be for Australia with a focus on the macroeconomic adjustments that would take place in response to the epidemic. Thus, we take an economy-wide approach to estimating the economic impacts of an H1N1 pandemic. Beutels et al. (2008), Lee and McKibbin (2004), and Smith et al. (2005) argue convincingly that economic analysis of public health emergencies of international concern (e.g., H1N1 influenza and SARS) should not take a partial equilibrium approach by focussing on only the health sector (or parts of the health sector) and forgone incomes resulting from disease-related morbidity and mortality, while ignoring effects in other parts of the economy (e.g., Sander et al. 2009). Illness and death due to public health emergencies raises perceptions of risk and leads to risk-modifying behaviour in an effort to reduce the risk of contracting illness, e.g., prophylactic absenteeism from work and public gatherings. Risk-modifying behaviours affect consumption and reduce labour productivity. Deaths due to illness reduce the availability of workers. Both of these effects will affect all parts of the economy to a greater or lesser extent. Further, Lee and McKibbin (2004), and Smith et al. (2005) show that the effects on the non-health sectors due to SARS and antimicrobial resistance are larger than the effects on the health sector. Thus, an economy-wide

¹ To quote WHO (2010b), "The reported number of fatal cases is an under representation of the actual numbers as many deaths are never tested or recognized as influenza related."

² These figures are as at September 2009.

approach is the ideal framework for properly evaluating the economic impacts of public health emergencies such as H1N1 influenza.

Our economy-wide analysis applies the MONASH-Health model of the Australian economy. MONASH-Health is a detailed, dynamic, computable general equilibrium (CGE) model of the Australian economy. The theoretical structure of MONASH-Health is similar to that of the MONASH model of Australia (Dixon and Rimmer 2002). MONASH-Health places special emphasis on the health sector to aid economic analysis of health sector issues. The health sector detail allows us to carefully target the increased demand on health services that an H1N1 epidemic would be expected to cause, while the general equilibrium nature of the model allows us to capture the indirect effects of the epidemic on the non-health sectors.

In applying the MONASH-Health model to analyse the economic effects of a H1N1 pandemic, we deviate from previous work in three important respects. First, three separate tourism sectors are identified in the model: domestic, inbound and outbound. Previous experience with the SARS epidemic indicates that international tourism is strongly negatively affected by epidemics of this kind (Pine and McKercher 2004; Wilder-Smith 2006). Identifying international tourism separately in the model allows us to target the likely negative effects on international tourism of a flu pandemic accurately. Second, MONASH-Health is modified for quarterly periodicity; quarterly behaviour is an uncommon characteristic of CGE models. This modification is important because pandemics tend to be of short, sharp duration. An annual model tends to smooth out short-term effects leading to potential underestimation of disruption. Third, MONASH-Health has the real world feature of inertia in the labour market (sticky real wages) to which we add the complementary feature of non-instantaneous price responses in the physical capital market (excess capacity). This allows us to avoid the assumption of full capacity utilisation (common in CGE models) in the presence of a demand contraction (such as a pandemic) that leads to the prediction of a strong export upturn in the short-run; Dixon and Rimmer (2010) show that this is an unrealistic response to a demand-contracting shock. Incorporating both quarterly periodicity and excess capacity represents a major development for CGE modelling in that it captures important short-run, real world macroeconomic mechanisms. This makes our framework ideal for assessing how an influenza pandemic affects the macroeconomy.

Applying the MONASH-Health model, we simulate the economic effects of two H1N1 epidemics in Australia: the 2009 outbreak and a significantly more severe episode. To represent the epidemiology of the two episodes we apply the classic Susceptible-Exposed-Infected-Removed (SEIR) model of infectious disease transmission (Kermack and McKendrick 1927; Anderson and May 1992). Our method is to calibrate the SEIR model on the 2009 epidemic and predict the effect on the economy as a whole using MONASH-Health. Based on the underlying characteristics of the 2009 epidemic from the SEIR model, we predict the effect of a stronger epidemic (with a higher number of infections) on the economy as a whole, taking account of its direct impact on health care, labour markets and the consumption of non-health goods as well as the indirect effects through macroeconomic variables including capital flows and the exchange rate.

There are a number of previous studies focussing on the economy-wide effects of global pandemics: some of these studies use macroeconomic (i.e., single sector) models. These include Fan (2003), Jonung and Roeger (2006) and Keogh-Brown et al. (2010). These studies have the advantage of applying quarterly models that allow them to capture the short, sharp nature of pandemics; they have the disadvantage of a single-sector approach that ignores sectors that are

particularly relevant to the study of the economic effects of epidemics (e.g., medical services, inbound and outbound tourism). Other studies apply a CGE (i.e., multi-sector) approach. These include Lee and McKibbin (2004), Chou et al. (2004) and McKibbin and Sidorenko (2006). While these studies apply models that have the advantage of identifying multiple sectors in the economy (albeit with a simple aggregated health sector), they are of annual periodicity and so are unable to capture the short, sharp nature of pandemics accurately. Dixon et al. (2010) are unique in applying a model with the dual advantage of identifying multiple sectors and having quarterly periodicity in analysing the effects on the United States of a serious H1N1 epidemic. We build on Dixon et al.'s framework by applying a model that incorporates multiple sectors, quarterly periodicity as well as a detailed representation of the health sector.

2. The nature of influenza pandemics

2.1 Overview

Detailed epidemiologic observations of influenza in human populations span more than 100 years, including four well-documented pandemics since the beginning of the 20th century: 1919, 1957, 1968 and 2009. The majority of influenza morbidity and mortality is due to seasonal strains that circulate each winter in temperate climates and over longer periods in the tropics (Nicholson et al. 2003). Less frequently, the emergence of antigenically novel viruses, often through cross-species transfer or reassortant events, can result in devastating outbreaks with large numbers of excess deaths in a single season (Doherty et al. 2006). The relative absence of immunity to such pandemic strains results in potential for widespread infection with higher attack rates (i.e., infection rates) across the age spectrum, and a greater burden of disease borne by otherwise healthy young adults than in inter-pandemic years (Ahmed et al. 2007). Following the first wave of infection, the new pandemic virus typically replaces previously circulating seasonal strains to produce annual epidemics in an increasingly immune population (Spicer and Lawrence 1984), with historical records describing variable patterns of subsequent disease. In 1918-19, multiple waves of infection occurred in a single year, some out of season, while in 1968, more severe second than first waves were observed in some populations (Fox and Kilbourne 1973).

Precise estimation of death rates due to influenza is made difficult by the potential for misclassification of cause of death. For example, this bias arises when deaths occur in the community prior to medical consultation, or result from some other diagnosed cause such as a cardiac event that might have been precipitated by underlying influenza (Warren-Gash et al. 2009). Having recovered from their infection, individuals are less likely to be reinfected with related H1N1 strains, and may retain some measure of broad cross-protection against unrelated seasonal influenza viruses (Steel et al. 2010).

2.2 How influenza pandemics affect human behaviour

Direct economic effects of illness resulting from influenza include increased healthcare expenditures by patients and funders (e.g., governments, insurers), and increased workloads for healthcare workers. Indirect effects include a smaller labour supply due to deaths, and increased

absenteeism from work by sick workers and by workers wishing to reduce the risk of contracting illness in the workplace, i.e., prophylactic absenteeism.³

Prophylactic absenteeism is one example of voluntary risk-modifying behaviour in response to a pandemic. Other examples are reduced domestic and international travel, and reduced public gatherings at sporting and other events (CBO 2006). Non-voluntary risk-modifying behaviour may be imposed on workers with children by school closures intended to mitigate the spread of the virus (Beutels et al. 2008). Thus, some workers will be forced to take leave to care for young children. Workers who take paid leave from work, whether forced or voluntary, reduce their firm's labour productivity, i.e., output per worker, unless other workers can fully replace output lost due to absenteeism. This may be difficult during an influenza pandemic because the virus will be widespread and while many workers may not present to the health system, they are likely to be less productive than would otherwise be the case.

It is unclear what attitudes firms have towards absenteeism during pandemics, including whether they prepare for such events or whether it affects their hiring behaviour. A related question is whether firms utilise workers differently during pandemics, e.g., do they expect present workers to work harder or longer to compensate for output lost due to absent workers? And do pandemics directly impact investment behaviour by firms? These issues have not been given much attention by pandemic researchers up to now.

Fan (2003) asserts that a pandemic will reduce business investment due to increased uncertainty and risk, leading to excess capacity. Similarly, consumer confidence will decline due to uncertainty and fear, leading to reduced spending as people elect to be homebound to reduce the probability of infection—this is another example of risk-modifying behaviour. Reduced consumer confidence may particularly affect services involving face-to-face contact (e.g., tourism, transportation and retail spending). James and Sargent (2006) argue that evidence from past pandemics suggests that it is mainly discretionary spending (e.g., tourism and transportation) that is reduced.

Fan (2003) also asserts that an epidemic does not need to be of high morbidity and mortality in order to exert a large psychological impact on attitudes to risk. For instance, although the 2003 SARS epidemic was characterised by low morbidity and mortality, it did have a large psychological impact on attitudes to risk. Fan (2003) argues that this paradox can be explained by:

- modern communication technology that transmits information almost instantaneously at low (or zero) cost; and
- the lack of adequate medical information on SARS.

James and Sargent (2006) evaluate this argument by examining evidence from the SARS epidemic and agree that people did experience increased fear of infection, e.g., 50 per cent of surveyed respondents in Taiwan reported wearing a mask during the height of SARS (p. 22). Nevertheless, they argue that the evidence indicates that the only economic impact during the SARS epidemic was on air travel to affected locations and related impacts on accommodation. Keogh-Brown and Smith (2008) perform a retrospective analysis of the economic impact of the

³ James and Sargent (2006) argue that there is little evidence of prophylactic absenteeism during past influenza pandemics. This stands in contrast to survey results from Sadique et al. (2007) where European respondents suggest that 34% of workers would take prophylactic absenteeism measures in the event of an influenza pandemic.

2003 SARS epidemic and find that the economic effects were mainly but not exclusively centred on East Asian regions, and that the effects went beyond air travel and accommodation.

The above discussion serves to illustrate the unsettled nature of researchers' understanding of how influenza pandemics affect human behaviour.

3. The economic model

MONASH-Health is a detailed, dynamic, CGE model of the Australian economy that places special emphasis on the health sector to facilitate economic analysis of health sector issues. Its theoretical structure is similar to the MONASH model of Australia: MONASH is described in detail in Dixon and Rimmer (2002). Below we provide a stylised description of MONASH-Health.

3.1 The health sector: treatments and services

MONASH-Health contains a detailed representation of the health sector: this represents a new development for CGE models. Previous work in this area usually depicts the health sector by subsuming it within a broader sector in either a multi-sector model (e.g., Keogh-Brown et al. 2009; McKibbin and Sidorenko 2006) or a single-sector model (e.g., Keogh-Brown and Smith 2010). A detailed representation of the health sector allows carefully targeted analysis of changes in (i) demand for health treatments caused by epidemics or health promotion activities, and (ii) supply of health treatments from potential changes in institutional arrangements and other health-sector reforms.

In representing the health sector, 18 health treatments (e.g., cardiovascular disease) are distinguished from six health commodities (e.g., pharmaceuticals) and services (e.g., hospitals); see Table 1. The health treatment activities are based on the International Classification of Diseases–10th Revision and their absolute and relative sizes in the model data are determined using statistics from AIHW (2004). The health (commodities and) services facilitate the provision of treatment activities and are health services that are typically recognisable in detailed national accounts data. Thus, the treatment activities (or industries) employ constant-returns-to-scale technology using a Leontief (fixed proportions) combination of health services. Each health treatment industry uses a unique combination of health services; this information is also sourced from AIHW (2004). Some treatment activities are pharmaceutical intensive (e.g., respiratory system diseases; diabetes mellitus); others are hospital intensive (e.g., injuries; neonatal). Dental services are mainly applied in oral treatments. Leontief production functions fix the pattern of health services usage by the health treatment industries unless there are changes in treatment technologies. The health treatment industries do not directly demand primary factors (land, labour and capital) but are linked to other sectors of the economy via their demands for health services as intermediate inputs. Thus, their demand for primary factors is indirect via the demand for primary factors by the industries that produce their intermediate inputs: health services (Table 2, column 1).

Health treatments are purchased by households as consumption (Table 3): these purchases are made at subsidised prices (65%) financed by government revenue. We make the limiting assumption that health treatment exchanges take place in a perfectly competitive market and all demands by households are met. In reality, demands are usually rationed (usually via queuing) due to either funding restrictions or capacity constraints. Further work is required to improve this

representation of the model; however, we do not feel this limiting assumption impacts the application of the model to assessing an H1N1 pandemic. Households have limited possibilities to substitute across health treatments if relative prices change: the own-price elasticities range from -0.4 to -0.8. Expenditure elasticities are mostly greater than 1.

Table 1 Health treatments and health services in MONASH-Health

<u>Health treatments</u>		<u>Health commodities and services</u>
1. Cardiovascular disease	2. Genitourinary	1. Human pharmaceuticals
3. Nervous system	4. Endocrine, nutritional and metabolic	2. Hospitals, nursing homes
5. Musculoskeletal	6. Skin diseases	3. Medical services
7. Injuries	8. Maternal conditions	4. Dental services
9. Respiratory system diseases	10. Infectious and parasitic diseases	5. Optometry
11. Oral health	12. Diabetes mellitus	6. Ambulance services
13. Mental disorders	14. Neonatal causes	
15. Digestive system	16. Congenital anomalies	
17. Neoplasms	18. Signs, symptoms, ill-defined conditions and other contact with the health system	

Table 2 Input-output structure in MONASH-Health (2009 \$A billion)

<u>Inputs</u>	<u>Industries</u>					<u>Total^a</u>
	(1) Health treatments	(2) Health services	(3) Primary	(4) Manufacturing	(5) Services	
Health treatments	0	1	0	1	7	9
Health services	70	1	0	1	1	74
Primary	0	0	19	65	16	101
Manufacturing	0	6	19	108	205	339
Services	0	10	17	55	588	669
Primary factors	0	55	72	94	695	916
Total ^a	70	74	127	325	1,512	2,107

Source: MONASH-Health database.

^a Totals may not sum due to rounding.

Table 3 Sales structure in MONASH-Health (2009 \$A billion)

Commodities	(1) Consumption	(2) Investment	(3) Government	(4) Exports	(5) Imports	Total ^a
Health treatments	24	0	0	3	0	26
Health services	7	0	0	0	-7	-1
Primary	17	7	1	89	-23	90
Manufacturing	184	93	0	142	-227	192
Services	382	177	132	72	-62	702
Total ^a	614	277	132	305	-319	1,009

Source: MONASH-Health database.

^a Totals may not sum due to rounding.

The health services sectors are treated in a fashion that is more typical of CGE models (e.g., Francois and Reinert 1997). There is a representative firm for each sector that employs

constant-returns-to-scale technology using a CES (constant elasticity of substitution) combination of primary factors and intermediate inputs. There is no substitution between the primary factor composite and intermediate inputs, and a CES of 0.5 between individual primary factors; intermediate inputs are used in fixed proportions. Regardless, health service industries mainly use primary factors as inputs and this is dominated by labour inputs (Table 2, column 2). Their outputs are almost exclusively sold to the health treatment industries (Table 2, column 1). A small amount of health services is sold to the representative household (Table 3, column 1); like the sales of health treatments, these sales are also made at prices that receive a 65% subsidy.

Integrating the health treatment, health services and non-health industries within a single, consistent framework allows us to capture the indirect effects of changes in demand for, or supply of, the treatment industries simultaneously with the direct effects of any change. This is the major advantage of using an economy-wide model with a health sector focus for analysing the economic impacts of health sector changes. Nevertheless, the output of the health treatment industries does not directly impact upon the performance of workers via changes in their health status. Current development of MONASH-Health is exploring such links.

3.2 The non-health sectors

MONASH-Health also contains 35 non-health sectors; Tables 2 and 3 present data on these sectors as broad aggregates. The non-health sectors use their own outputs as inputs as well as primary factors. The non-health sectors in MONASH-Health are treated similarly to the health services sectors: there is a representative firm for each sector that employs constant-returns-to-scale technology using a CES combination of primary factors and intermediate inputs. Substitution possibilities across all production inputs mirror those already described for the health services sectors. In contrast to the health services sectors, non-health firms' outputs can be sold to other firms, capital creators (for investment), the representative household, the government, or exported (Table 3). All domestic agents can choose to buy domestically-produced or imported goods; this choice is also handled by a CES function. Ex-duty prices of imports are assumed to be fixed. Exports are assumed to respond to foreign currency prices, which are endogenous. Thus, the terms of trade are endogenous and the economy is treated as 'almost small'. Total household consumption is assumed to be a fixed proportion of household disposable income; total government consumption is assumed to be fixed.

3.3 Dynamic mechanisms

MONASH-Health includes three types of dynamic mechanisms: capital accumulation; liability accumulation; and lagged adjustment processes. Capital accumulation is specified separately for each industry. An industry's capital stock at the start of year $t+1$ is its capital at the start of year t plus its investment during year t minus depreciation. Investment during year t is determined as a positive function of the expected rate of return on the industry's capital.⁴ Liability accumulation is specified for the public sector and foreign accounts. Public sector liability at the start of year $t+1$ is public sector liability at the start of year t plus the public sector deficit incurred during year t . Net foreign liabilities at the start of year $t+1$ are net foreign

⁴ In this application expected rates of return are only a function of current period variables, i.e., capital creators are assumed to have adaptive expectations. We feel this is appropriate for the application analysed here; an H1N1 epidemic is an event rather than a policy change. Further, it is a new event of unknown magnitude at the time it occurs. It is difficult to envisage how capital creators could accurately predict future variables affecting the rate of return given these characteristics of the epidemic.

liabilities at the start of year t plus the current account deficit in year t plus the effects of revaluations of assets and liabilities caused by changes in price levels and the exchange rate. Lagged adjustment processes are specified for the response of wage rates to gaps between the demand for and the supply of labour by occupation.⁵ Thus, unemployment is endogenous.

3.4 Model solution

The model is represented by equations specifying behavioural and definitional relationships. These can be compactly written in matrix form as

$$A\mathbf{v} = \mathbf{0}, \quad (1)$$

where A is an matrix of coefficients, \mathbf{v} is a vector of percentage changes (or changes) in model variables and $\mathbf{0}$ is a null vector. Model variables are divided into two mutually exclusive lists, exogenous and endogenous, thus closing the model. Many of the functions underlying (1) are highly nonlinear. Writing the equation system like (1) allows us to avoid finding the explicit forms for the nonlinear functions. It also allows us to minimise computational burdens by applying a derivative form of (1) when solving the model; this follows the method pioneered by Johansen (1960). Although the derivative form of (1) is linear, accurate solutions are generated by applying multistep solution procedures; see Dixon and Rimmer (2002), pp. 113–18 for more details.⁶

In a MONASH-Health simulation of the effects of a pandemic, we run the model twice to create the baseline and policy runs. The baseline is intended to be a plausible forecast while the policy run generates deviations away from the baseline caused by the pandemic. For the non-health treatment sectors, the baseline incorporates trends in industry technologies, household preferences, trade and demographic variables (see Dixon and Rimmer 2002, p.38, for further details). For the health treatment sectors, the baseline incorporates forecasts that have been developed from AIHW data outlining expected future demand (quantities) for individual treatments (Begg et al. 2007). Most macroeconomic variables are exogenous in the baseline so that their paths can be set in accordance with forecasts made by expert macroeconomic forecasting groups (e.g., Australian Treasury). This requires endogenisation of various macroeconomic propensities, such as the average propensity to consume, which then adjust to accommodate the exogenous paths for the macroeconomic variables.

In the policy run, macroeconomic variables must be endogenous. Correspondingly, macroeconomic propensities are exogenised and given the values they had in the baseline. More generally, all exogenous variables in the policy run have the values they had in the baseline with the exception of the variables of interest. Comparison of results from the policy and baseline runs then gives the effects of moving the variables of interest away from their baseline values. For the present study, the baseline and policy runs differ with regard to the values given to exogenous variables representing an outbreak of H1N1 influenza. We interpret the differences between the results in the baseline and the policy runs as the effects of the outbreak.

⁵ This last dynamic mechanism gives MONASH-Health New Keynesian behaviour with respect to the labour market.

⁶ The model is implemented and solved using the multistep algorithms available in the GEMPACK economic modelling software (Harrison and Pearson 1996).

3.5 Model modifications

Below we describe important model modifications relating to quarterly periodicity and capital idling. The modifications relating to adding three tourism sectors to the model (domestic, international inbound and outbound) are described in Verikios et al. (2010), section 3.5.

3.5.1 Quarterly periodicity

MONASH-Health typically produces annual results (e.g., Brown et al. 2009). But for this work the model has been modified to produce quarterly results. The modification is important because past influenza pandemics have had sharp effects over a short period (e.g., 3-6 months). An annual model tends to smooth out such effects leading to potential underestimation of disruption. For example, if an epidemic caused an 80% loss of inbound international tourism within a particular quarter, then the adjustment path of the tourism industry would be quite different from that in a situation in which international tourism declined by 20% for a year. Similarly, a 20% increase in a single quarter in demands for medical services related to infectious diseases would place more stress on the medical system than a 5% increase spread over a year. Fan (2003), Jonung and Roeger (2006) and Keogh-Brown et al. (2010) all apply models with quarterly periodicity to analyse economy-wide impacts of pandemics.

Annual CGE models like MONASH-Health are commonly solved in a recursive manner.⁷ Dynamic-recursive models usually divide time into discrete intervals and economic variables are assumed to change at the end of each interval. Such models take the form

$$Y = G(X), \quad (2)$$

where Y and X are the levels of the endogenous and exogenous variables in a period. Computations are then carried out according to

$$\Delta Y = G'(X) \Delta X, \quad (3)$$

where Δ refers to changes from one period to the next. If we have changes from one year to the next for the exogenous variables, i.e., ΔX , then to model quarterly changes we must divide these changes by four. Other changes required to move a discrete-time model from annual to quarterly periodicity are:

- a) equations must be added that handle quarterly accumulation of stock variables;
- b) the base data for the initial values of lagged variables must be altered to reflect quarterly rather than annual lags; and
- c) parameter values in equations describing partial adjustment mechanisms must be altered to reflect quarterly rather than annual adjustment.

We briefly describe (a) and (c) below; see Verikios et al. (2010), section 3.3, for further details.

Stock-flow relationships

Discrete time CGE models with annual periodicity usually have an equation explaining end-of-year (y) capital stocks KE_j^y (for the j -th industry) as a function of beginning-of-year capital stocks KB_j^y , and investment I_j^y and depreciation in the current year. Movements in KB_j^y

⁷ The exceptions are intertemporal models that compute results simultaneously for all time periods (e.g., McKibbin and Wilcoxon 1999; Malakellis 2000).

are set to reflect the annual growth rate of the capital stock in the initial solution (i.e., the initial data). I_j^y will usually be a function of rates of return on capital. We add an equation explaining end-of-quarter (q) capital stocks KE_j^q as a function of beginning-of-quarter capital stocks KB_j^q , and investment I_j^q and depreciation in the current quarter. Movements in KB_j^q reflect the quarterly growth rate of the capital stock in the initial solution, and $I_j^q = I_j^y/4$. Thus, the relationship between rates of return on capital and investment is still an annual one even though the periodicity of the model is now quarterly. This assumes that firms still make investment plans over a one year time horizon but only one quarter of those plans come online in the current quarter. Thus, KE_j^y and I_j^y are never realised, they are only planning variables.

Similar changes to those described above for the capital accumulation relationships are also made to the stock-flow relationships for debt, credit and equity.

Partial adjustment mechanisms

MONASH-Health contains an equation that controls the deviation of employment from the baseline whereby it is assumed that in policy simulations the deviation in the real wage from the basecase level increases at a rate that is proportional to the deviation in the gap between employment and labour supply from its basecase level:

$$\left\{ \frac{W^t}{W_b^t} - 1 \right\} = \left\{ \frac{W^{t-1}}{W_b^{t-1}} - 1 \right\} + \beta \left\{ \frac{E^t}{E_b^t} - \frac{LS^t}{LS_b^t} \right\} + U^t, \forall t. \quad (4)$$

In (4), W^t and W_b^t are the real wage rates in year t in policy and baseline simulations, E^t and LS^t are employment and labour supply in year t in the policy simulation, E_b^t and LS_b^t are employment and labour supply in year t in the baseline simulations, U^t is a slack variable set exogenously at zero, and β is a positive parameter.

The relationship between real wage and employment deviations from baseline is controlled by β . If β is zero, the real wage stays at its baseline level regardless of labour market conditions in the current period; if β is one, the real wage responds flexibly to clear non-zero excess demand for labour in the current period. β is usually calibrated so that the employment deviations of a shock to the economy are approximately zero after about five years, e.g., $\beta = 0.5$.⁸ In a quarterly model we wish this relationship to continue to hold. So we divide the parameter by four so that the employment deviations of a shock to the economy are approximately zero after about twenty quarters, e.g., $\beta = 0.125$.

3.5.2 Capital idling

The discussion in Section 2.2 noted the importance of risk-modifying behaviour during pandemics that leads to reductions in demand (e.g., tourism and transportation). Previous analysis has found reductions in demand to be an important part of the negative shock to the economy from pandemics, e.g., Keogh-Brown et al. (2010). But these analyses have assumed full capacity utilisation despite the fact that variation in capacity utilisation is a prominent feature

⁸ This labour market assumption is consistent with conventional macroeconomic modelling in which the NAIRU is exogenous. It is also compatible with search models and efficiency-wage theory.

of recessions in Australia (Otto 1999), the US (BGFRS 2010), and most likely other developed economies. Moreover, Otto (1999) provides evidence that demand shocks explain nearly all of the variation in capacity utilisation for Australia. This suggests that modelling capacity utilisation may be important in analysing the effects of demand-contracting events. This view is supported by Dixon and Rimmer (2010) in their analysis of the contemporaneous US recession using a dynamic CGE model.

Dixon and Rimmer (2010) show that assuming full capacity utilisation in each industry involves sharp reductions in rental rates on capital in response to reductions in demand. Lower rental rates on capital lead to a net capital outflow and a real exchange rate depreciation; this drives an export upturn in the short run. This is an unrealistic response to a demand-contracting shock: for example, this is not a feature of the current US recession. Such unrealistic export responses can only be moderated by allowing for less-than-full capacity utilisation.

Variable capacity utilisation has been extensively analysed in various contexts within the real business cycle (RBC) literature (Nakajima 2005). In allowing for excess capacity here, we follow Dixon and Rimmer (2010). Their representation of idle capital is most closely related to the RBC model of Cooley et al. (1995) who represent production by a continuum of firms that each period decide whether to run a plant or not. That is, adjustment in capital used occurs along the extensive margin. The basic idea in Dixon and Rimmer (2010) is that capital rental rates are sluggishly adjusting mark-ups on variable costs (wages plus materials) that fall in response to excess capacity; whereas the typical view of rental rates is that they represent market-clearing prices for the use of existing capital stocks. We implement the former view in the policy run and this requires that we distinguish between capital in use (KU_j^t) and capital in existence (KE_j^t): excess capacity is where $KE_j^t > KU_j^t$. Thus, capital accumulation relationships are written in terms of KE_j .

We allow for sticky rental rates and excess capacity in the policy run only, via

$$\left\{ \frac{Q_j^t}{Qb_j^t} - 1 \right\} = \left\{ \frac{Q_j^{t-1}}{Qb_j^{t-1}} - 1 \right\} + \alpha \left\{ \frac{KU_j^t}{KE_j^t} - 1 \right\} + S_j^t, \quad \forall t; \quad (5)$$

where Q_j^t and Qb_j^t are the rental rates for industry j in quarter t in policy and baseline simulations, S_j^t is a slack variable, and α is a positive parameter. S_j^t is used to implement (5) in the policy run only, and to turn off (5) once $KE_j^t = KU_j^t$. Note also that $Q_j^t = f(KU_j^t, \dots)$, $\forall t$, where f is a decreasing function.

In (5) the degree of rigidity in the rental price deviation $\left\{ \frac{Q_j^t}{Qb_j^t} - 1 \right\}$ is controlled by α ,

which determines how responsive the rental price deviation is to excess capacity per quarter: it is set at 0.05. This means that if we set all other terms to zero in (5), 10% excess capacity will cause a -0.5% rental price deviation from baseline. This gives rental prices a high degree of downward rigidity in the presence of excess capacity, which with all else constant will work to remove excess capacity quickly once demand conditions begin to move towards baseline levels.

Two adjustments are also made to the investment function. First, expected rates of return on investment, $EROR_j^t$, will be lower under excess capacity than under full capacity utilisation by defining them as

$$EROR_j^t = \left\{ \frac{KU_j^t}{KE_j^t} \right\} ROR_j^t - \left\{ 1 - \frac{KU_j^t}{KE_j^t} \right\} D_j, \quad \forall t. \quad (6)$$

That is, $EROR_j^t$ will be a weighted average of the rate of return on capital, ROR_j^t , and the negative of the depreciation rate D_j , where the weights are the share of capital in existence that is in use and the share not in use. So we are assuming that capital not in use earns no rental and deteriorates at the depreciation rate.

Second, demands for additional capacity in quarter t (when demand for capital exceeds supply during recovery) are partially met by recommissioning idle capital from period $t-1$. The appropriate parameter values are set so that around 16.5% of idle capital is recommissioned per quarter to satisfy demands for additional capacity; this is equivalent to two thirds of idle capital being recommissioned per year.

4. Influenza scenarios

The earlier discussion of previous analyses of pandemics and their potential economic effects flagged a number of channels through which an economy could be affected by a serious outbreak of H1N1 influenza. Considering these channels, we decide on four types of economic shocks to impose on MONASH-Health to simulate an H1N1 pandemic:

- (1) a surge in demand for hospital and other medical services;
- (2) a temporary upsurge in sick leave and school closures requiring withdrawal of parents from the labour force;
- (3) some deaths with a related permanent reduction in the labour force; and
- (4) temporary reductions in inbound and outbound international tourism and business travel.

We have developed two quantitative scenarios covering factors (1) to (3), to represent the ‘first wave’ of infection due to a newly emerged pandemic influenza strain; (4) is determined separately below. The scenarios are both constructed from the classic Susceptible-Exposed-Infected-Removed (SEIR) model of infectious disease transmission (Kermack and McKendrick 1927; Anderson and May 1992) with allowance for unobserved (mild and non-presenting) transmission and pre-existing immunity in a portion of the population. The number of people in the population who are susceptible (S), exposed (E), infectious (I) or recovered (R) are described by a set of coupled nonlinear first-order differential equations and associated initial conditions:

$$\begin{aligned} \frac{dS}{dt} &= -\beta IS, \\ \frac{dE}{dt} &= \beta IS - \alpha \gamma E, \end{aligned}$$

$$\frac{dI}{dt} = \alpha\gamma E - \nu I ,$$

$$\frac{dR}{dt} = \nu I ;$$

where β is the production rate of newly-exposed individuals, γ is the transition rate from the exposed to infectious class (and so $1/\gamma$ is the average duration spent in the exposed class), ν is the transition rate from infectious to recovered ($1/\nu$ is the average duration of infectiousness) and α is the proportion of all infections that present to health service providers (i.e., the proportion of infectious individuals that are *not* mild or non-presenting). The initial conditions are $S(0) = (1 - z)N$, $E(0) = 0$, $I(0) = 10$ and $R(0) = zN$, where N is the population size and z is the proportion of the population immune to infection prior to the onset of the pandemic.

The infection rate, driving the S to E transition, is proportional to the product of S and I and so the model is nonlinear. Given an initial reproduction number (the number of secondary cases arising from a primary case, given by β/ν) that is greater than 1, the model will naturally show an initial exponential increase in the proportion of the population that is infected. The epidemic peaks when the reproduction number is equal to 1 (i.e., when $S(t) = \nu/\beta$), and then declines due to exhaustion of the susceptible pool, returning to a steady state ($I = 0$). The initial reproduction number is tuned to provide the desired total number of infections over the course of the epidemic. The time scale of the epidemic is set by adjusting the serial interval $1/\gamma + 1/\nu$, which is the average time between infection of one individual and subsequent infection of the next.

4.1 Scenario 1: the 2009 H1N1 outbreak

Our method is to calibrate the SEIR model on the 2009 epidemic and predict the effect on the economy as whole using MONASH-Health. Thus, the first scenario is calibrated, wherever possible, on available data for the 2009 H1N1 outbreak as at October 2010 (DHA 2009).⁹ A number of serosurveys (Gilbert et al. 2010, McVernon et al. 2010) covering the Australian population indicate that approximately 11% of the Australian population were infected in the course of the 2009 Australian pandemic,¹⁰ which is higher than the values used for planning purposes in the *Australian Health Management Plan for Pandemic Influenza* (DHA 2008). Twelve per cent of the population were assumed to be protected prior to the outbreak, based on serological data (McVernon et al. 2010). The 2.2 million Australians infected with H1N1 experience symptoms over 2009:2–2009:4 (i.e., quarters 2–4 of 2009). The dynamics of the outbreak meant that 87% of all new infections occurred in 2009:3; the shocks to MONASH-Health described below reflect these dynamics. In formulating these shocks we have deliberately chosen conservative assumptions that give smaller economic disruptions. We feel this is appropriate given the unsettled nature of researchers’ understanding of how influenza pandemics

⁹ DHA (2009) is our source for data available as at October 2010. This data was first published during 2009 but was subsequently revised in 2010 after the end of the pandemic.

¹⁰ We include the following multiplicative factors to allow for the influenza-like illness presentation rate to differ from the true H1N1 rate: 5 for general practitioners and flu clinics, and 2 for non-ICU hospital admissions. These factors were chosen based on expert opinion and consultation with government during the early phases (May–June 2009) of the Australian outbreak (see Table 3).

affect human behaviour. Given the scenario parameters, we assume the following shocks to the economy.

(1) That out of the 2.17 million people who experience symptoms: 1.96 million seek no medical attention but spend \$5 (2009 dollars) on pharmaceuticals; 0.21 million seek medical attention (by visiting a general practitioner or a flu clinic) but are not hospitalised, incurring expenses of \$61 (2009 dollars); 4,305 are hospitalised and survive, incurring expenses of \$3,564 (2009 dollars); 700 are hospitalised and spend time in an ICU, incurring expenses of \$85,395 (2009 dollars), of which 506 survive and 194 die.^{11, 12} The presentation venues for the 2.17 million symptomatic individuals have been chosen to reflect the official Australian presentation counts. Table 4 shows the assumed proportional split of influenza-like illness (ILI) presentation to different venues, and the values are calibrated to reproduce the 2009 Australian experience. Altogether medical expenses are \$100 million (2009 dollars) incurred over May–December 2009. This amounts to a 6.3% increase in demand for medical services relating to respiratory treatments over 2009:2–2009:4. In MONASH-Health, respiratory treatments are mainly composed of four inputs: human pharmaceuticals, hospitals and nursing homes, medical services, and ambulance services. Thus, it is these services whose demand will be mostly affected by the increased demand for respiratory treatments. We assume that demand for respiratory treatments returns to normal in 2010:1.

(2) That workers miss a total of 1.44 million workdays over 2009:2–2009:4 on account of their own sickness including 0.4 million days while caring for children who are either sick or kept home by school closures. This assumes: 0.5 workdays are lost per working age person who experiences symptoms and seeks no medical attention; 2.4 workdays are lost per working age person who seeks medical attention (by visiting a general practitioner or a flu clinic); 13.9 workdays are lost per working age person who is hospitalised. Working parents caring for homebound children are assumed to miss a comparable number of workdays except in the case of hospitalisation where half as many days are missed.¹³ Workdays lost by parents while caring for children are scaled for the share of families with all parents working (Australian Bureau of Statistics 2009). The total loss in workdays translates to a reduction in labour productivity of 0.22% (= 1.44 million days out of the 646 million days available from 10.77 million workers supplying 60 days each quarter). We assume that labour productivity returns to normal in 2010:1. Note that our workday losses assume neither prophylactic absenteeism nor lower productivity by workers who may not present to the health system but will be less productive than would otherwise be the case, both of which would increase the workday losses.

(3) That of the 194 persons who die, 78 are workers. This translates into a permanent reduction in the labour force of 0.0007% over 2009:2–2009:4.

¹¹ The per unit costs for each type of H1N1 case are determined as follows: pharmaceutical cost is representative of a generic over-the-counter symptomatic relief flu medication; general practitioner and flu clinic cost is the cost of a standard doctor visit in 2009; hospital cost is the cost of a standard hospital visit in 2009; ICU cost is based on estimates from Higgins et al. (2011).

¹² When the 2009 outbreak occurred the government announced an H1N1 vaccination program; our expenditure estimates do not include vaccination expenditure due to the lack of available data.

¹³ Per capita workday losses (for workers and parents) were estimated by Molinari et al. (2007) for working age people (18–64 years) in their study of seasonal influenza in the US; their estimates take account of workforce participation rates.

Table 4 Key parameter values for the simulated epidemics in scenarios 1 and 2

Parameter	Scenario 1	Scenario 2	Comment
Percentage of population infected with H1N1 and displaying symptoms	11	30	Scenario 1: parameter values are based, wherever possible, on estimates from the 2009 H1N1 pandemic in Australia (DHA 2009). Where data were unavailable, historical (20 th century) influenza pandemics, published pandemic preparedness modelling studies and expert opinion are used to inform the choices.
Percentage of H1N1 cases that are mild	95	75	
Percentage of H1N1 cases that are moderate	4	15	
Percentage of H1N1 cases that are severe	1	10	
Non-presenting, GP, flu clinic split for mild cases (%)	99, 0.5, 0.5	99, 0.5, 0.5	Scenario 2: parameter values are based on the assumption that the proportion infected with H1N1 is increased threefold relative to scenario 1 and by 50% relative to the primary scenario considered in the <i>Australian Health Management Plan for Pandemic Influenza</i> (DHA 2008).
Non-presenting, GP, flu clinic split for moderate cases (%)	90, 5, 5	90, 5, 5	
Percentage of severe cases allocated to GP, flu clinic, hospital (non-ICU), ICU	37.5, 37.5, 21.5, 3.5	32, 33, 30, 5	Scenario 1: splits to hospital and ICU are set to reflect actual recorded presentations. Scenario 2: splits are skewed to reflect more hospitalisations.
Percentage of ICU admissions resulting in death	28	56 (a further 20% of hospitalised cases die relative to scenario 1)	Scenario 1: as given in DHA (2009). Scenario 2: 0.26% of the Australian population dies, a value consistent with the case-fatality-rate in the Western world during the 1918-19 influenza pandemic.
Total ILI cases per true H1N1 case at GP, flu clinic, hospital and ICU (multiplicative factor)	5, 5, 2, 1	5, 5, 2, 1	Based on early findings of a surge in ILI (non-H1N1) presentations to these venues, presumably due to heightened public awareness and concern.

(4) We assume that during the three peak quarters of infection and cost that inbound and outbound tourism are negatively affected. Here we follow previous estimates of the effects of SARS and the Iraq war on Australian tourism for 2003 by Dwyer et al. (2006). Given that these estimates are for both the effects of SARS and the Iraq war, we conservatively assume half the effects estimated by Dwyer et al. (2006), i.e., during the first two peak quarters of infection (2009:2–2009:3) that inbound tourism is negatively affected by 9.2% and 2.2%. For outbound tourism (including business travel), we think it reasonable to posit that some potential Australian travellers would be dissuaded from international travel by fears of becoming symptomatic on vacation. Thus, we assume that the outbound tourism effect is half that of the inbound effect. Tourism numbers stay at their recessed levels in 2009:4 and then recover smoothly to their baseline levels over 2010:1–2010:2. Australians who cancel their outbound tourism redirect their purchases to other goods. This is an optimistic assumption; a pessimistic assumption is that consumers save their money until the pandemic ceases.

Note that part of the shocks for scenario 1, in terms of increased demand for medical services and absenteeism, are not too dissimilar to the expected shocks that would occur every

year due to influenza and ILIs. So the true impact on demand for medical services and absenteeism *may* be somewhat less than we have predicted.

4.2 Scenario 2: a severe H1N1 outbreak

Based on the underlying characteristics of the 2009 epidemic from the SEIR model, we predict the effect of a stronger epidemic (with a higher number of infections) on the economy as a whole. Thus our second scenario is based on a severe H1N1 outbreak assumed to occur during the same period as scenario 1, i.e., 2009:2–2009:4, and similar to the more severe scenarios considered in modelling work that informed the *Australian Health Management Plan for Pandemic Influenza* (AMHPPI). The proportion infected with H1N1 is increased to 30% of the population or 6 million individuals, a 50% increase over the baseline case assumed in the AMHPPI and a three-fold increase over that observed in 2009. To account for non-H1N1 ILIs, the total number of symptomatic infections is 8.1 million. Given the scenario parameters, we assume the following shocks to the economy.

- (1) Out of the 8.1 million people who experience symptoms: 5.3 million seek no medical attention but spend \$5 (2009 dollars) on pharmaceuticals; 2.6 million seek medical attention (by visiting a general practitioner or a flu clinic) but are not hospitalised, incurring expenses of \$61 (2009 dollars); 180,000 are hospitalised and survive, incurring expenses of \$3,564 (2009 dollars); 64,000 are hospitalised and spend time in an ICU, incurring expenses of \$85,395 (2009 dollars), of which 11,000 survive and 53,000 die (see Table 4 for the proportional breakdown of presentations). Altogether medical expenses are \$2.4 billion (2009 dollars) incurred over May–December 2009. This amounts to a 154% increase in demand for medical services relating to respiratory treatments during the period 2009:2–2009:4. We assume that demand for respiratory treatments returns to normal in 2010:1.
- (2) That workers miss a total of 7.9 million workdays over 2009:2–2009:4 on account of their own sickness and a further 3.8 million days while caring for children who are either sick or kept home by school closures. The total loss in workdays translates to a reduction in labour productivity of 1.8%. We assume that labour productivity returns to normal in 2010:1.
- (3) That out of the 53,000 persons who die, 21,000 are workers. This translates into a permanent reduction in the labour force of 0.19% over 2009:2–2009:4.
- (4) That inbound tourism falls by 39% over 2009:2–2009:4 and then recovers smoothly to its basecase level over the next four quarters. In setting the shocks for inbound tourism we considered the experience in Asia during the SARS epidemic of 2003. This episode suggests that regions suffering a widespread influenza infection could incur reductions in inbound tourism in the range of 20%–70% during the peak infection period (Pine and McKercher 2004; Wilder-Smith 2006). For our hypothetical severe H1N1 epidemic, we adopt a number towards the lower end of this range (39%). As in scenario 1, we assume that the outbound tourism (and business travel) effect is half that of the inbound effect over 2009:2–2009:4. Outbound tourism then recovers smoothly to its basecase level over the next four quarters. Australians who cancel their outbound tourism redirect their purchases to other goods.

Although both scenarios simulate a global H1N1 pandemic, we are unable to explicitly apply pandemic shocks to countries other than Australia in our model. This limitation may overstate or understate the extent to which Australia's international trade in goods and assets is affected by the pandemic and the size of the impacts on economic activity. We cannot overcome

this limitation within the present framework but we do flag the importance of this limitation in the discussion of our results.

5. Results

5.1 Scenario 1: the 2009 H1N1 outbreak

The main effects of the 2009 outbreak occur in 2009 and peak in 2009:4, by which time GDP and employment are 0.9% and 0.7% below baseline (Table 5); the size of the deviations over 2009:2–2009:4 reflect the dynamics of the pandemics in terms of new infection rates per quarter. The decline in GDP relative to employment reflects the loss in labour productivity. On average through 2009, the epidemic reduces GDP and employment by 0.5% and 0.4%. Through 2010, GDP is a little lower even though employment is unchanged. This reflects the slow return to full capacity after the pandemic ends; thus capital in use is 0.2% below baseline through 2010.

Table 5 Scenario 1 effects (percentage deviation from baseline)

Variable	2009				2010				
	Q2	Q3	Q4	Average	Q1	Q2	Q3	Q4	Average
GDP	-0.3	-0.8	-0.9	-0.5	-0.4	0.0	0.1	0.1	-0.1
Employment	-0.3	-0.6	-0.7	-0.4	-0.4	0.1	0.2	0.1	0.0
Capital in use	-0.3	-0.7	-0.9	-0.5	-0.4	-0.1	-0.1	-0.1	-0.2
Capital in existence	0.0	0.0	0.0	0.0	0.0	-0.1	-0.1	-0.1	-0.1
Investment	-0.2	-1.2	-2.1	-0.9	-1.9	-0.6	0.1	0.1	-0.5
Consumption	-0.3	-0.6	-0.8	-0.4	-0.4	-0.1	0.0	0.0	-0.1
Exports	-1.0	-1.5	-1.1	-0.9	0.4	0.5	0.2	0.2	0.3
Imports	-0.7	-1.3	-1.5	-0.9	-0.8	-0.1	0.1	0.1	-0.2

The epidemic reduces investment by 0.9% through 2009. In 2009:2 investment falls below baseline (-0.2%) because demand-contracting (tourism) and cost-increasing (labour productivity) shocks reduce the rental value of capital. This damps expected rates of return and thereby reduces investment. In 2009:2 the epidemic causes excess capacity to appear in some industries (Figure 1), particularly those related to tourism and construction. Excess capacity in 2009:2 has a strongly negative effect on investment in 2009:3. Weak investment in 2009:3 causes further excess capacity to appear, explaining weak investment in 2009:4. In 2010:1, much of the pick up in demand for capital associated with the recovery in labour productivity and the start of the recovery in tourism is satisfied by working down the excess capacity that appeared in 2009:2–2009:4. With excess capacity peaking in 2009:4, investment starts to move back towards baseline in 2010:1. This is because excess capacity in 2010:1 is declining as capital in existence adjusts down and capital in use adjusts up. By 2010:2, excess capacity is eliminated.

Through 2011, Figure 1 shows average deviations for GDP and employment of 0.0% and 0.1%. The epidemic-related reduction in employment in 2009 causes real wage rates to be lower than they otherwise would have been. This allows the Australian economy to arrive in 2010 with enhanced international competitiveness so that when tourism recovers and the other epidemic-related shocks disappear, employment and output move above their baseline values. The improvement in Australia's international competitiveness predicted here is likely to be tempered by a global H1N1 pandemic that affects Australia's trading partners and competitors, i.e., their

real wage rates are also likely to move below baseline, but most likely with different dynamics. This would make Australia's exports and domestically-produced goods less competitive than suggested by our results here. It is difficult to account for these trading partner and competitor effects in a national model like the one we apply here.

Figure 1 indicates the relative importance of the different shocks in determining the overall employment effects. We see that H1N1 deaths have an almost imperceptible effect on aggregate employment due to their small number. Increased expenditure on health services and away from other items of household expenditure has slightly larger employment effects: employment is a little higher in the short run. This is because production of medical services is considerably more labour intensive than production of most other items of household expenditure. Extra medical expenditures in 2009 have a small negative effect on aggregate employment in 2010. This is a reflection of the wage mechanism mentioned earlier: extra employment in 2009 associated with health expenditures weakens the competitive advantage that the Australian economy experiences in 2010. We see that the main drivers of lower employment are lost workdays and the fall in international tourism, with each effect contributing roughly equally to the overall employment loss of 0.8%.

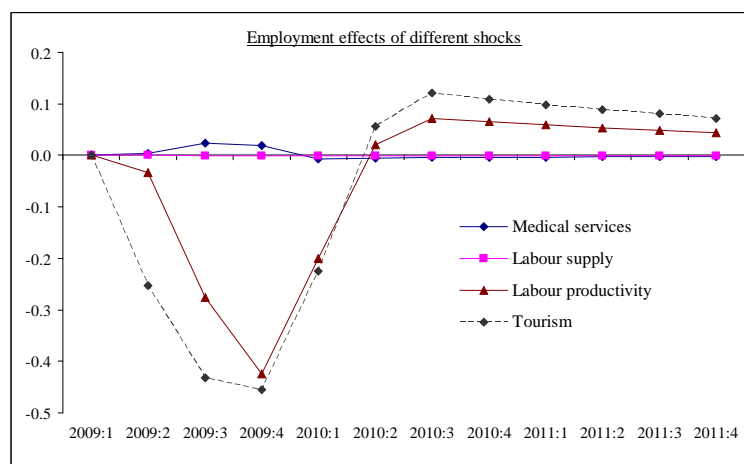
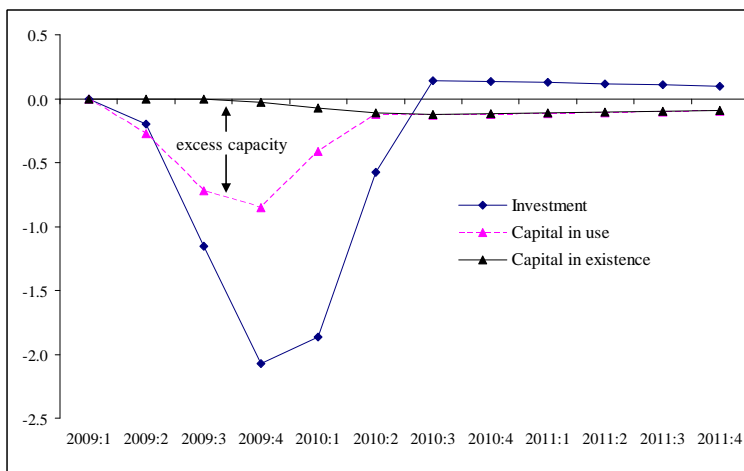
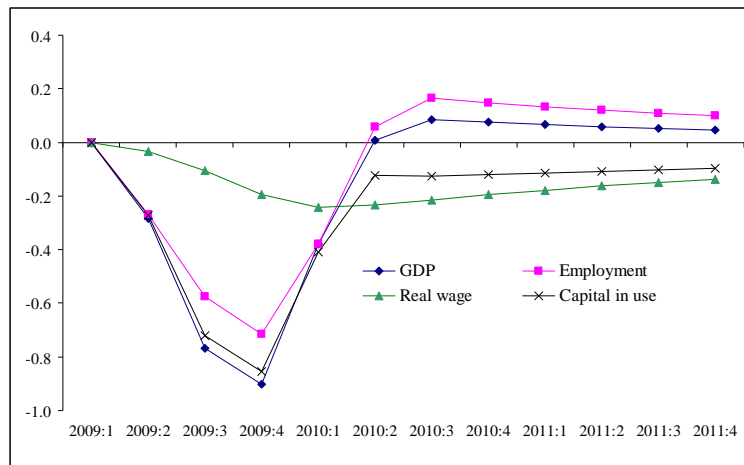
Figure 2 compares our results for scenario 1 with the typical CGE assumption of full capacity utilisation (FCU); the differences in GDP effects are stark. Dropping the assumption of variable capacity utilisation (VCU) reduces the peak GDP effect from -0.9% to -0.02%. As discussed in Section 3.5.2, assuming FCU involves sharp reductions in rental rates on capital in response to reductions in demand. Lower rental rates on capital lead to a net capital outflow and a real exchange rate depreciation; this drives an export upturn in the short-run. We see this mechanism at play in Figure 2 where under FCU the real exchange rate¹⁴ depreciates initially and stays below baseline through 2009 and 2010, compared to almost no initial depreciation with VCU. Thus, exports fall less with FCU. Although exports decline sharply by 2009:3 with VCU, reflecting the 8% reduction in inbound tourism, exports begin to recover by 2009:4 even though inbound tourism does not fully recover until 2010:2. The recovery of exports is assisted by the real exchange rate depreciation, associated with the peak effect on investment, that begins in 2009:4.

5.2 Scenario 2: a severe H1N1 outbreak

The severe outbreak has similar dynamics to the 2009 outbreak, and so its peak effect also occurs in 2009:4. Importantly, however, the effects are much larger with GDP and employment 6.2% and 4.1% below baseline in 2009:4 (Table 6). These effects are between six and seven times larger than the peak effects of the 2009 outbreak. This is a combination of two major differences between the two outbreaks: (i) the clinical attack rate is three times higher in the severe outbreak; and (ii) the proportion of infected cases that are moderate or worse is five times higher in the severe outbreak. On average through 2009, the severe outbreak reduces GDP and employment by 3.6% and 2.2%. Through 2010, the economy recovers strongly but is still recessed; GDP and employment are 0.7% and 0.4% below baseline. As observed in the 2009 outbreak, the recovery is impeded by a slow return to full capacity after the pandemic ends. The slow return to full capacity is impeded here by the slow recovery in tourism numbers through 2010.

¹⁴ The real exchange rate is measured by movements in the Australian price level compared with price levels in trading partners expressed in a common currency. Negative movements in the real exchange rate indicate improvements in the international competitiveness of the Australian economy.

**Figure 1 Scenario 1 effects on selected aggregates
(percentage deviations from baseline)**



**Figure 2 Scenario 1 effects with variable and full capacity utilisation
(percentage deviations from baseline)**

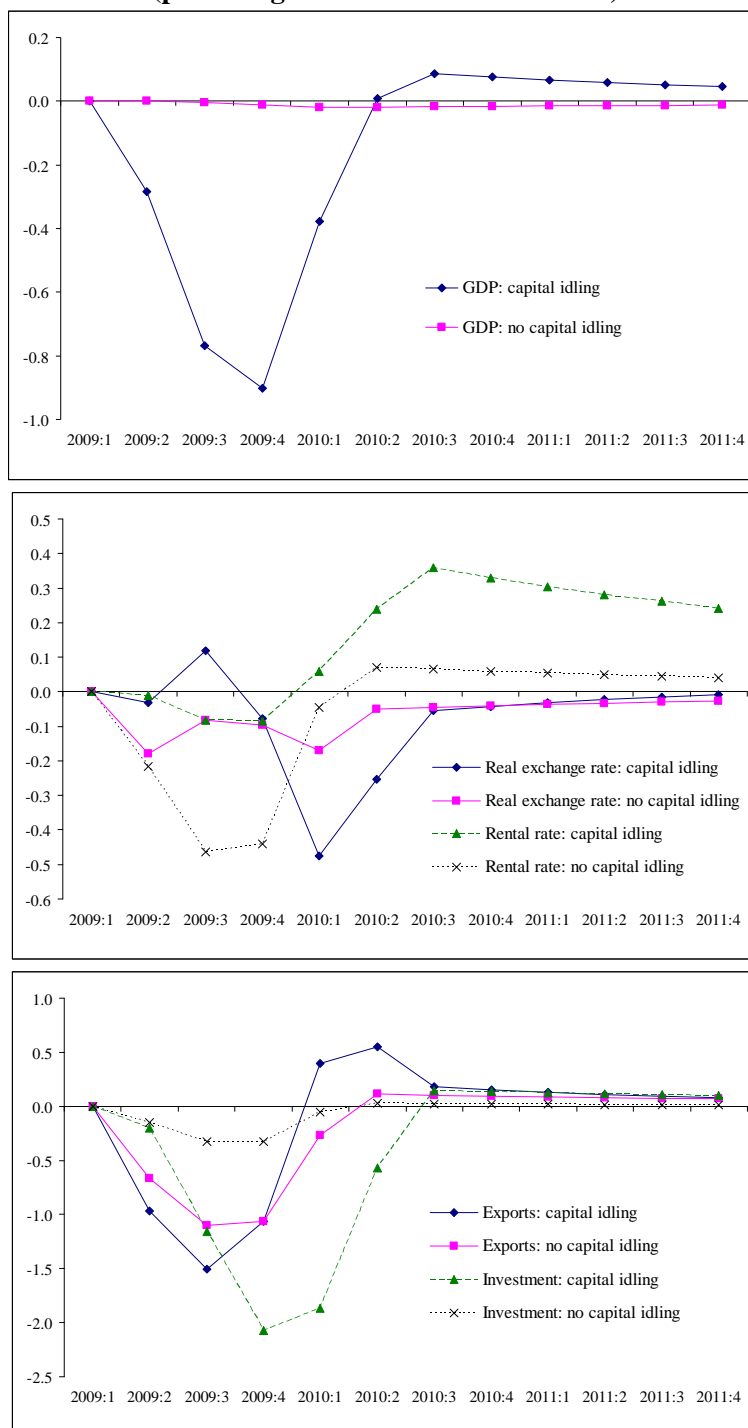


Table 6 Scenario 2 effects (percentage deviation from baseline)

Variable	2009				2010				
	Q2	Q3	Q4	Average	Q1	Q2	Q3	Q4	Average
GDP	-2.6	-5.7	-6.2	-3.6	-2.6	-0.5	0.2	0.2	-0.7
Employment	-1.3	-3.4	-4.1	-2.2	-2.6	-0.2	0.6	0.7	-0.4
Capital in use	-2.5	-5.5	-5.8	-3.5	-2.7	-1.0	-1.0	-0.9	-1.4
Capital in existence	0.0	0.0	-0.3	-0.1	-0.6	-0.8	-0.9	-0.9	-0.8
Investment	-1.9	-8.8	-14.6	-6.3	-12.2	-4.2	0.3	0.6	-3.9
Consumption	-1.8	-4.4	-5.0	-2.8	-2.8	-0.7	0.0	0.1	-0.9
Exports	-6.2	-9.2	-5.8	-5.3	2.4	1.5	0.1	0.5	1.1
Imports	-3.0	-6.8	-8.1	-4.5	-5.1	-1.9	-0.3	0.4	-1.7

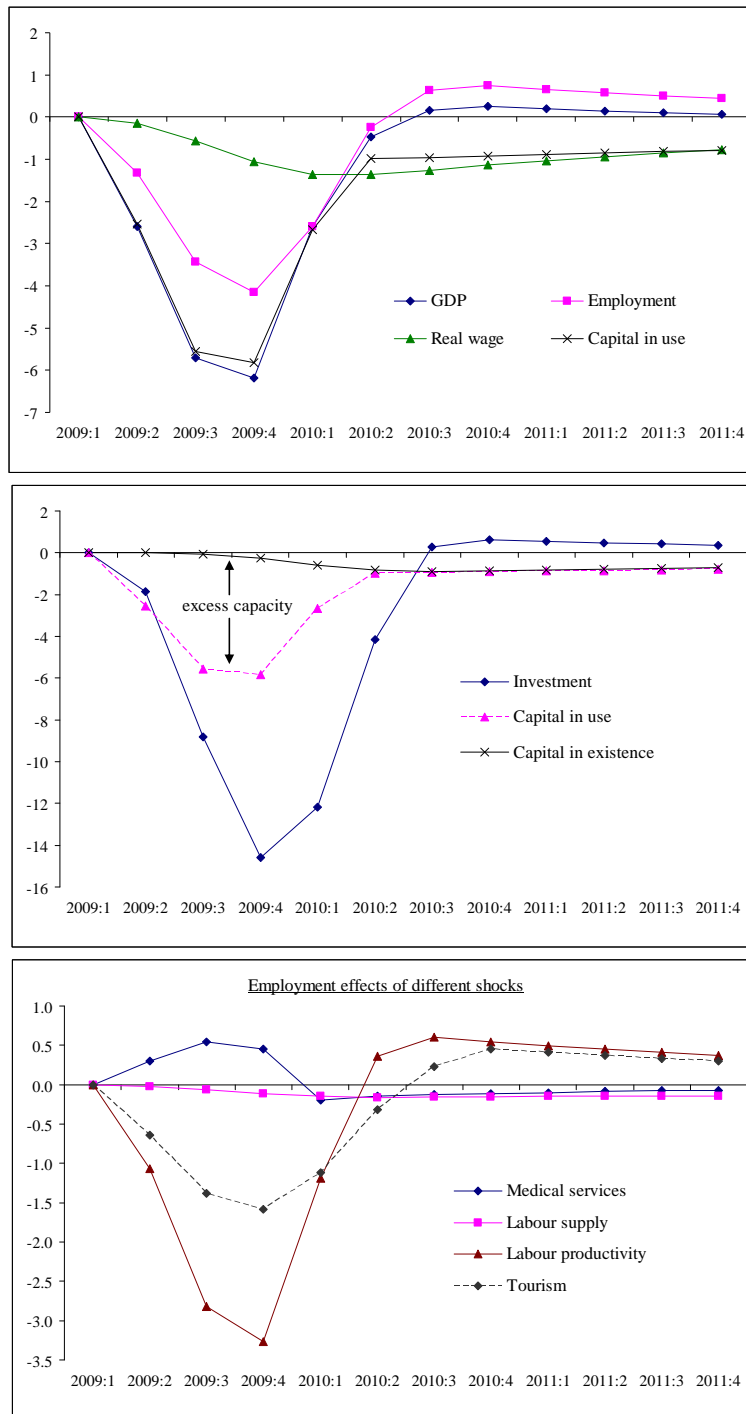
An important contributor to the larger effects in the severe outbreak is the much greater peak effect on investment: -14.6% versus -2.1% in 2009:4. With a much larger fall in investment, a much larger level of excess capacity emerges in 2009 (Figure 3). Like investment, excess capacity peaks in 2009:4. From 2010:1, investment starts to move back towards baseline reflecting the beginning of the pickup in capital in use once the pandemic ends. At the same time, capital in existence continues to adjust down. Excess capacity is eliminated by 2010:3 and so investment moves above baseline.

Comparing the relative importance of the different shocks in determining the overall employment effects (Figure 3), we see that H1N1 deaths have almost no effect on employment in the short run but have a small negative effect in the long run. Short-run results in MONASH-Health are mainly demand driven and the deaths have little effect on aggregate demand. In the longer run, employment is determined mainly by labour supply: demand for labour adjusts to changes in supply via wage movements. Beyond 2010, H1N1-related deaths in 2009 reduce employment by reducing labour supply. Increased expenditure on health services and away from other items of household expenditure has similar effects in the severe outbreak to the 2009 outbreak: a positive effect on employment in the short run; a small negative effect on aggregate employment in 2010.

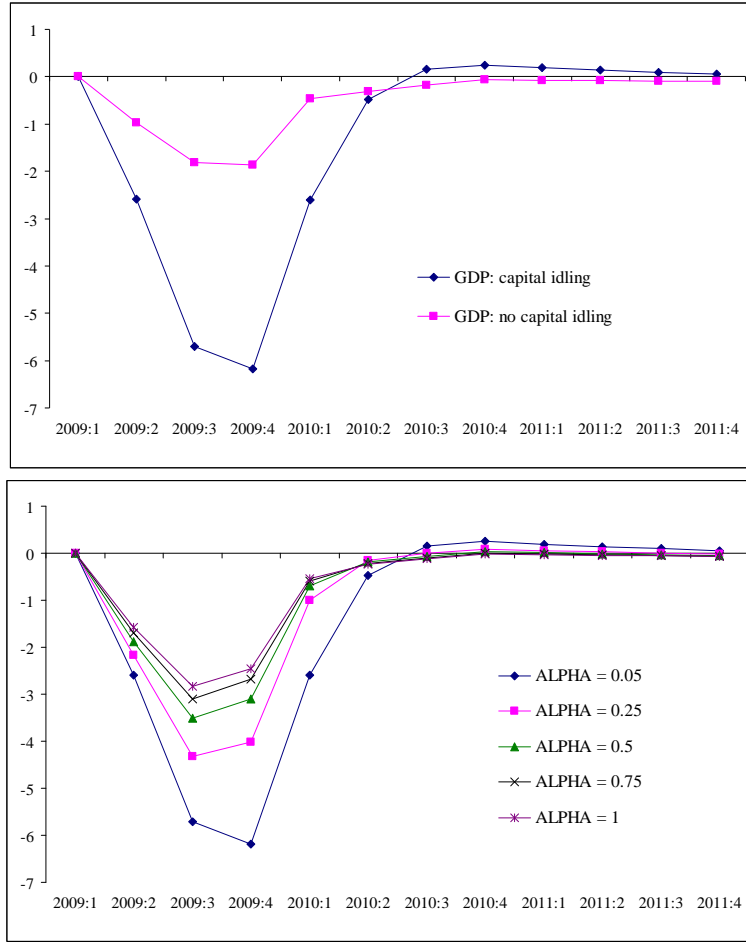
Similar to scenario 1, the main drivers of lower employment are lost workdays (labour productivity) and the fall in international tourism, but now lost workdays are much more employment-reducing than lower tourism; 3.3% versus 1.6% in 2009:4. This reflects the assumption that the risk-modifying behaviour leading to lower tourism is not assumed to increase in direct proportion to infection rates as we move from the 2009 outbreak to the severe outbreak. It is not obvious whether this is an appropriate assumption. Nevertheless, as the simulated effects are approximately linear in the shocks, we know that larger tourism effects would lead to larger adverse employment effects.

Figure 4 shows, as in scenario 1, that the assumption of VCU or FCU leads to very different GDP deviations from baseline. Assuming VCU gives a peak GDP effect of -6.2% in 2009:4; assuming FCU gives a peak GDP effect of -1.9%. The mechanism driving this difference has been outlined earlier. The relative differences in peak GDP effects between the VCU and FCU simulations for scenario 2 are much smaller than those observed for scenario 1. This suggests that the degree of rental price stickiness may be important in determining the depth of the trough in economic activity when a pandemic hits (see the parameter α in equation (5)). We test this assumption by varying α from its current value of 0.05 through to 1 (Figure 4). We see that increasing the value of α reduces the size of the GDP effect but by decreasing amounts.

**Figure 3 Scenario 2 effects on selected aggregates
(percentage deviations from baseline)**



**Figure 4 Scenario 2 GDP effects with variable and full capacity utilisation
(percentage deviations from baseline)**



5.3 Systematic sensitivity analysis

We are able to test the sensitivity of the results to variations in α and other MONASH-Health parameters systematically. Table 7 reports estimated means and standard deviations for GDP effects from scenario 2 if the relevant parameters vary symmetrically within a chosen range and follow a triangular distribution. The calculation of means and standard deviations was carried out using the systematic sensitivity methods automated in the GEMPACK economic modelling software (Harrison and Pearson 1996). These methods rely on a Gaussian quadrature to select a modest number of different sets of values for the varying parameters (DeVuyst and Preckel 1997). The model is solved using each different set of parameter values and the means and standard deviations are calculated over the several different solutions of the model. The calculated means and standard deviations will be good approximations to the true means and standard deviations provided that: (i) simulation results are well approximated by a third-order polynomial in the varying shocks and parameters; (ii) varying shocks and parameters have a symmetric distribution; (iii) shocks and parameters do not both vary at once; and (iv) shocks and parameters either have a zero correlation or are perfectly correlated within a specified range chosen by the user (Arndt and Pearson 1996).

Table 7 Systematic sensitivity analysis of GDP effects in scenario 2 (percentage deviation from baseline)

	2009				2010		
	Q2	Q3	Q4	Q1	Q2	Q3	Q4
1. Mean	-2.6	-5.7	-6.2	-2.6	-0.5	0.2	0.2
2. Standard deviation from variation in:							
a. Rental price stickiness ^a	0.20	0.68	1.16	1.02	0.26	0.08	0.09
b. Real wage stickiness ^{b, c}	0.06	0.23	0.44	0.41	0.21	0.11	0.08
c. Primary factor substitution ^c	0.05	0.09	0.06	0.13	0.11	0.07	0.05
d. Export demand elasticity ^c	0.19	0.32	0.12	0.15	0.10	0.02	0.00

^a This is α in equation (5). It is varied in the range $\alpha/20$ and $\alpha*20$. ^b This is β in equation (4). ^c Varied in the range $\pm 50\%$.

Table 7 presents the estimated means and standard deviations for GDP with respect to symmetric, triangular variations in parameters. Row 1 presents the calculated mean across the different solutions and, as expected, it is the same as for the original simulation as reported in Table 6. The other results in Table 7 report the values of the standard deviations as each parameter is varied; most parameters are varied by $\pm 50\%$ (see notes to Table 7). The results indicate that our GDP estimates are robust with respect to variations in the elasticity of primary factor substitution (row 2c) and the elasticity of export demands (row 2d). In contrast, the GDP effects are rather sensitive to values of the parameters determining the speed with which disequilibrium is eliminated in the capital market (row 2a) and labour market (row 2b), i.e., the estimated standard deviations are not insignificant compared to the size of the model results. This confirms the *ad hoc* sensitivity analysis of the rental price stickiness conducted in Section 5.2.

The sensitivity of the GDP effects to rental price and real wage stickiness does vary over 2009:2–2010:4. The peak effects (2009) are not so sensitive to the degree of rental price and real wage stickiness that we cannot be confident of the sign of the GDP effects. This is not true in the year following the peak effects (2010) where the means are only two standard deviations or so away from changing sign. The greater sensitivity of the results in 2010 for rental price and real wage stickiness is due to the importance of these assumptions in determining the rebound in economic activity above baseline once the pandemic ends. This is demonstrated in Figures 2 and 4, which compare the FCU and VCU results for each scenario. Assuming FCU (i.e., no rental price stickiness) removes the rebound above baseline in both scenarios. Assuming no real wage stickiness would have a similar influence. The sensitivity of the model results to these two important parameters should be noted when considering our results. Nevertheless, it should also be noted that both excess capacity and real wage stickiness are real-world phenomena for developed economies. Thus, the empirical issue to be determined is the degree of inertia in the markets for physical capital and labour rather than their existence.

6. Discussion

Our results show that the possible GDP effects on Australia of a mild H1N1 pandemic (scenario 1) are in the range -0.5% to 0% in the peak year, depending on the degree of capacity utilisation assumed. For a severe H1N1 pandemic, our results range from -3.6% to -1.2%. The results are not directly comparable to most previous studies and, as far as we are aware, there are

no studies that have estimated the economic effects of the 2009 H1N1 pandemic for Australia or other countries. There are a number of studies that have analysed past influenza pandemics and hypothetical future influenza pandemics. Most recently, Dixon et al. (2010) estimate the economic effects of a serious H1N1 epidemic for the US; their work showed a GDP effect of -1.6% in the peak year. Keogh-Brown et al. (2010) estimate the effects on the UK of the 1957 or 1968 influenza pandemics could affect GDP by -0.58% in the peak year, and a more extreme scenario could increase this to -4.5% or -6%. The estimates for both of these studies are strongly driven by the lost workdays and reduced consumption by households; this is also true for our analysis. Jonung and Roeger (2006) conduct a similar study to Keogh-Brown et al. (2010) but estimate the effects of mild and severe influenza pandemics on the European economy: their estimates of GDP effects in the peak year range from -1.1% to -4%. McKibbin and Sidorenko (2006) estimate the global effects of a broad range of pandemic influenzas. For Australia they estimate GDP effects of -0.8% to -10.6%.

Our peak year GDP effects are within the range of estimates of previous studies cited above. The lower bound of our estimates is smaller than the lowest estimate of the previous studies, e.g., 0% versus -0.58%. Moreover, the upper bound of our estimates is only one-third as large as the highest estimate of previous studies, e.g., -3.6% versus -10.6%. There are differences across all these studies including ours. In terms of the analytical framework, differences relate to periodicity, sectoral detail, and capacity utilisation. In terms of the scenarios modelled, differences relate to infectiousness (infection rates), virulence (death rates) and timing. Further differences relate to the nature of the shocks imposed for a given scenario, e.g., the degree of risk-modifying behaviour undertaken in response to the pandemic. This last set of differences is legitimate as researchers have well-grounded reasons for assuming different degrees of risk-modifying behaviour and the response by authorities. Our approach to estimating pandemic responses is conservative and we have adopted responses toward the middle of the range of previous studies due to the uncertain nature of the response by people to pandemics. This approach is reflected in the fact that our estimates of economic disruption are low compared to previous studies.

7. Concluding remarks

We apply a quarterly CGE model to simulate the economic effects on the Australian economy of two H1N1 epidemics; the relatively mild 2009 outbreak and a more severe episode infecting about 8 million Australians (i.e., around one third of the population). The use of a model with quarterly periodicity rather than the usual annual periodicity allows the analysis to capture the short-run nature of influenza pandemics. Such an event would have its initial economic effects concentrated over no more than one or two quarters. We also deviate from previous work in this area by incorporating the real-world phenomenon of excess capacity, and we demonstrate its importance when simulating a demand-contracting shock like an influenza pandemic.

Our analysis shows that an H1N1 pandemic could have significant macroeconomic effects. It is likely that it would reduce household demands for international travel and demands by international tourists for hotels, travel and other services within Australia. It is also likely that industries would face increased costs via absenteeism. Both the demand decreases and cost increases associated with an epidemic of the size assumed here could be expected to cause a sharp reduction in investment with resulting adverse effects on employment and GDP. The

simulations show reductions in GDP and employment in the peak quarter of 0.9% and 0.7% for the 2009 epidemic and 6.2% and 4.1% for a severe epidemic. Nevertheless, the size of these effects is highly dependent on the assumed degree of capacity utilisation and labour market inertia. The analysis also demonstrates that the sharp downturn in economic activity would be short-lived with employment and GDP returning to business-as-usual growth rates within two to four quarters, depending on the severity of the epidemic.

A major contribution of this work is that it represents the first attempt to estimate the economic effects on Australia of an H1N1 pandemic. As far as we are aware, the only previous work of this kind is for the US (Dixon et al. 2010) that found significant short-run macroeconomic effects from an H1N1 epidemic: our results are consistent with this finding. Incorporating both quarterly periodicity and excess capacity also represents a major development for CGE modelling is that it captures important short-run, real world macroeconomic mechanisms. This makes our framework ideal for assessing how an influenza pandemic affects the macroeconomy. Nevertheless, it should be noted that our analysis does not take into account a number of responses that are difficult to quantify but are likely as the severity of the epidemic increases, such as: the reaching of capacity constraints for hospitals in general and intensive care units in particular; the change in risk-modifying behaviour as deaths become a much larger proportion of population; and the ameliorating effects of policy responses such as vaccination, prophylactic anti-viral medications and their costs.

Our work points to a number of policy implications for governments, who wish to find the best means to minimise the health and social costs of future influenza epidemics, and for businesses, who wish to minimise their potential loss of productivity. Our results demonstrate that the macroeconomic effects of an influenza epidemic similar to, or moderately more severe than, the 2009 H1N1 epidemic in Australia are significant, but are also likely to be short lived. The largest economic impacts of an influenza epidemic are driven by lost workdays, whether as a result of illness or formal social distancing measures to contain the epidemic such as school and workplace closure; this is consistent with the work of Keogh-Brown et al. (2009, 2010). These findings demonstrate the importance of ongoing appraisal of the severity of an evolving health emergency to ensure a proportionate and beneficial national policy response (Bishop et al. 2009). These findings further highlight an existing evidence gap regarding optimal application and feasibility of stringent distancing measures (even over short periods), with realistic estimation of likely impacts on disease transmission (Leung and Nicoll 2010). Additionally, the benefits of private sector preparedness to maintain continuity are reinforced, including protecting workers' health through appropriate infection control and flexible work arrangements, and planning for contingency such as the need for childcare arrangements in the event of school closure (DIISR 2006). In all, this evaluation suggests that in order to minimise the health and economic impact of emerging health threats such as pandemic influenza, there is a need for decentralised contingency planning, not just in the health sector, but across the range of activities potentially affected (Moss et al. 2011).

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